

## REMARKS

### STATUS OF THE CLAIMS

Claims 1-5, 16, 17, 19-22, 31, 33-38, 40-44, and 63, 65-67 were pending in this application. Claims 3-15, 18, 30-38, and 40-44 have been cancelled without prejudice. Claims 23-29, 45-62, and 64 have been withdrawn. Claims 1, 2, 16, 17, 19-22, 63, 65, and 67 have been amended. Following entry of the amendments claims 1, 2, 16, 17, 19-22, 63, and 65-67 will be pending and at issue.

### SUPPORT FOR AMENDMENTS TO THE CLAIMS

Claims 1 and 63 have been amended to include the phrases “obtaining an isolated polynucleotide that encodes said alpha-2B-adrenergic receptor, or a complement thereof, or a fragment thereof, or a complement of said fragment, that includes nucleotides 901 to 909 of SEQ ID NO: 1, or nucleotides 901 to 909 of SEQ ID NO: 2, or their complements” and “detecting in said isolated polynucleotide the presence or absence of a deletion polymorphism, said deletion polymorphism *exclusively consisting of* the deletion of nucleotide positions 901 to 909 of SEQ ID NO: 1” to more clearly define Applicants’ invention (emphasis added). Support for these amendments can be found throughout the specification as filed, e.g., pages 4-7, pages 10-11, pages 15-16, etc. Claims 1 and 63 have also been amended to include the phrase “establishing that a ligand-binding function of said alpha-2B-adrenergic receptor is reduced if said deletion polymorphism is present as compared to said ligand-binding function if said deletion polymorphism is absent” to more clearly define Applicants’ invention. Support for these amendments can be found throughout the specification as filed, e.g., page 9, lines 22-25, page 66, lines 13-28, page 67, lines 1-2, Table 2, page 63, etc. Other clarifying amendments have been made to the claims, as well, and support for these can be found throughout the specification as filed.

Claim 2 has been amended to include the phrase “said detecting comprises a hybridization step.” Support can be found in original claim 16, as filed. Claim 16 has been amended to depend from claim 1. Claims 17 and 19-22 have been amended to depend from

claim 2. Claim 65 has been amended to clarify its language and claim 67 has been amended to correct inadvertent and/or typographical errors.

These changes are believed not to introduce new matter, and their entry is respectfully requested. In making these amendments, Applicants do not concede that the subject matter of such claims or of any cancelled claims was in fact disclosed or taught by the cited prior art. Rather, Applicant reserves the right to pursue such protection at a later point in time and merely seeks to pursue protection for the subject matter presented in this submission.

#### **AMENDMENTS TO THE SPECIFICATION**

The specification has been amended to delete the GenBank Accession #AF316895 and replace it with the GenBank Accession #AF005900. The Examiner had identified in a previous Office Action of October 11, 2005 that the GenBank Accession #AF009500 included in the specification as filed was the GenBank record for *Streptococcus suis* 16S ribosomal RNA gene rather than the record for the human, wild-type alpha-2B-adrenergic receptor gene. In Applicants' response of April 11, 2005, Applicants amended the specification to replace GenBank Accession number #AF009500 with GenBank Accession number AF#316895 for the human alpha-2B-adrenergic receptor gene. In the Office Action of January 5, 2006, the Examiner objected to the specification and stated that this amendment introduced new matter since the "replacement of #AF009500 with #AF316895 is not the correction of an obvious error." *See* Office Action, pg. 2. Additionally, the Examiner noted that the publication date of GenBank Accession number # AF316895 is after the filing date of the present application. The Examiner further noted that GenBank Accession number # AF316895 includes the alpha-2B-adrenergic receptor gene with the 9-nucleotide deletion, rather than the longer, 1353 bp. wild-type alpha-2B-adrenergic receptor gene. Applicants respectfully thank the Examiner for pointing out this discrepancy.

In response, Applicants have amended the specification to replace GenBank Accession number #AF316895 with #AF005900 at the first paragraph on p. 14 and the last paragraph on p. 57 of the specification. GenBank Accession number #AF005900 correctly refers to the longer,

1353 bp. human, wild-type alpha-2B-adrenergic receptor gene. A copy of the GenBank record associated with number #AF005900 is attached. Applicant further submits that replacement of the original #AF009500 with new #AF005900 in the specification is simply the correction of a clear typographical error in which the “5” and the “9” in the Accession number were accidentally transposed in the present application. Thus, this is nothing more than correction of an “obvious error.” In addition, Applicant notes that the GenBank Accession number #AF005900 record has two versions, one of which dates back to 1997, before the filing of the specification and a newer version dating back to 2004. Both versions refer to the same human, wild-type alpha-2B-adrenergic receptor gene sequence (SEQ ID NO: 1) identified in the specification. Accordingly, Applicants respectfully request that Examiner reconsider and withdraw the objection to the specification.

#### **CLAIM OBJECTIONS**

Applicants have amendment claim 67 to correct the typographical error identified by the Examiner in which the word “phosphorylation” was misspelled. Applicants again thank the Examiner for identifying this error.

#### **REJECTIONS UNDER 35 U.S.C. § 112, SECOND PARAGRAPH**

Claim 19 was rejected under 35 U.S.C. § 112, second paragraph as allegedly indefinite. This rejection has been rendered moot in view of the amendments made to claim 16 and to the other claims. Thus, Applicants respectfully request that the Examiner withdraw this ground of rejection.

#### **REJECTIONS UNDER 35 U.S.C. § 112, FIRST PARAGRAPH**

Claims 31, 33-37, 38, and 40-44 were rejected under 35 U.S.C. § 112, first paragraph, as allegedly containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Without agreeing with the Examiner's rejection but to expedite prosecution of this application, Applicants have cancelled claims 31, 33-37, 38, and 40-44. Thus, Applicants respectfully request withdrawal of this ground of rejection.

#### **REJECTIONS UNDER 35 U.S.C. § 102**

Claims 31, 33-38 and 40-44 are rejected under 35 U.S.C. § 102(a) as allegedly being unpatentable over Heinonen ("The Identification of a Three-Amino Acid Deletion in the  $\alpha$  2B-Adrenergic Receptor that is Associated with Reduced Basal Metabolic Rate in Obese Subjects," Journal of Clinical Endocrinology & Metabolism, 84:2429-2433 (1999)). In view of the cancellation of claims 31, 33-38 and 40-44, this rejection has been rendered moot. Accordingly, Applicants respectfully request withdrawal of this ground of rejection.

Claims 1-5, 63, and 65-67 are rejected under 35 U.S.C. § 102(a) as allegedly being unpatentable over Jewell-Motz ("An Acidic Motif within the Third Intracellular Loop of the  $\alpha_2C2$  Adrenergic Receptor is Required for Agonist-Promoted Phosphorylation and Desensitization," Biochemistry, 34:11946-11953 (1995)). Applicant traverses this ground of rejection.

The Examiner stated that Jewell-Motz teaches a deletion or substitution of "a 16 amino acid stretch of glutamic acid residues from the alpha-2B-adrenergic receptor molecule" that would "inherently include instant SEQ ID NO: 3 which encodes three glutamic acid residues within a 16 amino acid repeat sequence of glutamic acids in the alpha-2B-adrenergic receptor molecule." See Office Action, p. 6. However, Jewell-Motz does not teach "detecting in said isolated polynucleotide the presence or absence of a deletion polymorphism, said deletion polymorphism exclusively consisting of the deletion of nucleotide positions 901 to 909 of SEQ ID NO: 1" of amended independent claims 1 and 63. Jewell-Motz teaches at most a deletion of a 16 amino acid stretch, but does not teach a polymorphic site "*exclusively consisting of the deletion of nucleotide positions 901 to 909 of SEQ ID NO: 1*" (emphasis added). The Examiner further agreed that "Jewell-Motz et al. do not teach a method wherein the polymorphic site

detected is limited to nucleotides 901 to 909.” *See* Office Action, p. 15. Thus, Jewell-Motz does not disclose all of the elements of the amended claims.

Accordingly, Applicants respectfully request withdrawal of this ground of rejection against independent claims 1 and 63, and the claims that depend therefrom and incorporate all of the elements of claims 1 and 63.

### **REJECTIONS UNDER 35 U.S.C. § 103**

Claims 16, 17, 20, and 22 are rejected under 35 U.S.C. § 103(a) as allegedly being unpatentable over either Heinonen in view of Newton (Chapter 6: Primers,” in *PCR Essential Data*, C.R. Newton, ed., John Wiley & Sons, Chichester, 1995, pp. 49-56). Applicant traverses this ground of rejection.

Three requirements must be met for a prima facie case of obviousness. First, the prior art references must teach all the limitations of the claims. Second, there must be a motivation to modify the reference or combine the teachings to produce the claimed invention. Third, a reasonable expectation of success is required.

The cited prior art references do not teach all of the elements of the claims. Heinonen does not teach “establishing that a ligand-binding function of said alpha-2B-adrenergic receptor is reduced if said deletion polymorphism is present as compared to said ligand-binding function if said deletion polymorphism is absent” of amended claim 1 (from which claim 16 has now been amended to depend). The instant application explains that the “major signaling phenotypes of the alpha-2BAR De1301-303 polymorphism is one of decreased agonist-promoted phosphorylation which results in a complete loss of the ability for the receptor to undergo agonist-promoted desensitization and *a decrease in receptor coupling*” (emphasis added). *See* p. 66, lines 13-17. In addition, the instant application indicates that physiologic consequences, such as a predisposition to salt-sensitive hypertension, could be related to either of these phenotypes. *See Id.* In contrast, Heinonen did not assign any specific cellular function to the polymorphism. Thus, Heinonen further did not establish that a ligand-binding function of said alpha-2B-

adrenergic receptor is reduced, as required by amended claim 16. Newton does not remedy this deficiency in Heinonen since Newton's teachings are focused on the design of primers for PCR.

Accordingly, the combination of Heinonen and Newton does not teach all of the elements of the claims, and so cannot render independent claim 16 obvious, nor the claims that depend therefrom (claims 17, 20, and 21). Therefore, withdrawal of this ground of rejection of claims 16, 17, 20, and 22 is respectfully requested.

Claims 19 and 21 are rejected under 35 U.S.C. § 103(a) as allegedly being unpatentable over either Heinonen in view of Newton (Chapter 6: Primers," in *PCR Essential Data*, C.R. Newton, ed., John Wiley & Sons, Chichester, 1995, pp. 49-56), and further in view of Snapir, et al. Applicant traverses this ground of rejection.

Claims 19 and 21 have been amended to depend from claim 2, which depends from claim 1. For at least the reasons explained above, Heinonen does not teach all of the elements of claim 1. Specifically, Heinonen does not teach "establishing that a ligand-binding function of said alpha-2B-adrenergic receptor is reduced." Again, Newton does not remedy this deficiency for at least the reasons stated above.

Accordingly, the combination of Heinonen and Newton does not teach all of the elements of the claims 19 and 21, and so cannot render independent claim 16 obvious. Therefore, withdrawal of this ground of rejection of claims 19 and 21 is respectfully requested.

Claims 1-5, 63, and 65-67 are rejected under 35 U.S.C. § 103(a) as allegedly being unpatentable over Jewell-Motz in view of Heinonen. Applicant traverses this ground of rejection.

The cited prior art references do not teach all of the elements of the claims. The Examiner explained that this 103 rejection is written against a "narrow interpretation of the rejected claims which would require the detection exclusively of polymorphic positions 901 to 909 of SEQ ID NO: 1 or 2 and not a polymorphic region of greater length," though the Examiner noted that the instant claims are "not so limited." See Office Action, p. 13 and p. 15. As explained above, independent claims 1 and 63 have been amended to recite "detecting in said isolated polynucleotide the presence or absence of a deletion polymorphism, said deletion

polymorphism *exclusively consisting of* the deletion of nucleotide positions 901 to 909 of SEQ ID NO: 1” (emphasis added). Thus, the instant claims are now limited as the Examiner suggested.

In addition, claims 1 and 63 have been amended to further recite “establishing that a ligand-binding function of said alpha-2B-adrenergic receptor is reduced if said deletion polymorphism is present as compared to said ligand-binding function if said deletion polymorphism is absent.” For at least the reasons stated above, Heinonen does not disclose this element of the claims. Heinonen did not assign any specific cellular function to the polymorphism, and thus further did not determine that the polymorphism caused a decrease in ligand binding of the receptor. Jewell-Motz does not remedy this deficiency. Jewell-Motz stated that the mutated receptors “bound [<sup>3</sup>H]yohimbine with the same affinity as was found for wild-type  $\alpha_2C2$ ” and found that the mutated receptors also “displayed the same affinities for the agonist epinephrine as compared to wild-type.” *See* Jewell-Motz, p. 11949. The binding affinities were “very similar” between the mutated and wild-type receptors, and so Jewell-Motz did not find that the deletion of the 16 glutamine residues caused a decrease in ligand binding of the receptor, as recited by the amended claims. *See Id.*

Accordingly, the combination of Jewell-Motz and Heinonen does not teach all of the elements of the claims, and so cannot render independent claim 1 and 63 obvious, nor the claims that depend therefrom (claims 2-5 and 65-67). Therefore, withdrawal of this ground of rejection of claims 1-5, 63, and 65-67 is respectfully requested.

## CONCLUSION

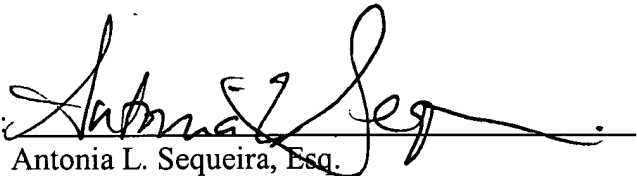
Withdrawal of the pending rejections and reconsideration of the claims are respectfully requested, and a notice of allowance is earnestly solicited. If the Examiner has any questions concerning this Response, the Examiner is invited to telephone Applicant's representative at (650) 335-7185.

Respectfully submitted,  
Kersten M. Small and Stephen Bryant Liggett

Dated: \_\_\_\_\_

5/5/04

By: \_\_\_\_\_



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[Comment](#) [Features](#) [Sequence](#)

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 AUTHORS Cayla, C., Schaak, S., Bouloumie, A., Devedjian, J.C. and Paris, H.  
 TITLE Alpha2C2-adrenergic receptor gene  
 JOURNAL Unpublished  
 REFERENCE 2 (bases 1 to 1353)  
 AUTHORS Cayla, C., Schaak, S., Bouloumie, A., Devedjian, J.C. and Paris, H.  
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 JOURNAL Submitted (29-MAY-1997) INSERM Unit 317, Institut Louis Bugnard, CHU Rangueil, Toulouse 31403, France  
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DEFINITION Homo sapiens alpha2B-adrenergic receptor (alpha2C2AR) gene, complete cds.

ACCESSION AF005900 REGION: 5500..6852

VERSION AF005900.2 GI:33439705

KEYWORDS

SOURCE Homo sapiens (human)

ORGANISM Homo sapiens

Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Primates; Haplorrhini; Catarrhini; Hominidae; Homo.

REFERENCE 1 (bases 1 to 1353)

AUTHORS

Cayla, C., Heinonen, P., Viikari, L., Schaak, S., Snapir, A., Bouloumie, A., Karvonen, M. K., Pesonen, U., Scheinin, M. and Paris, H.

TITLE

Cloning, characterisation and identification of several polymorphisms in the promoter region of the human alpha2B-adrenergic receptor gene

JOURNAL

Biochem. Pharmacol. 67 (3), 469-478 (2004)

PUBMED

15037199

REFERENCE 2 (bases 1 to 1353)

AUTHORS

Cayla, C., Schaak, S., Bouloumie, A., Devedjian, J. C. and Paris, H.

TITLE

Direct Submission

JOURNAL

Submitted (29-MAY-1997) INSERM Unit 317, Institut Louis Bugnard, CHU Rangueil, Toulouse 31403, France

REFERENCE 3 (bases 1 to 1353)

AUTHORS

Cayla, C., Heinonen, P., Viikari, L., Schaak, S., Snapir, A., Bouloumie, A., Karvonen, M., Pesonen, U., Scheinin, M. and Paris, H.

TITLE

Direct Submission

JOURNAL

Submitted (05-AUG-2003) INSERM Unit 317, Institut Louis Bugnard, CHU Rangueil, Toulouse 31403, France

REMARK

Sequence update by submitter

COMMENT On Aug 5, 2003 this sequence version replaced gi:2245627.

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